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Expert Scientific Panel on Chronic Wasting Disease

Dr. Trent Bollinger
CCWHC, SK, Canada

Dr. Peter Caley
CSIRO, Canberra, Australia

Dr. Evelyn Merrill
University of Alberta, AB, Canada

Dr. Fracois Messier
Chair, Univ. of Saskatchewan, SK, Canada

Dr. Michael W. Miller
Colorado Div. of Wildlife, CO, USA

See next page for additional authors

Authors

Dr. Trent Bollinger, Dr. Peter Caley, Dr. Evelyn Merrill, Dr. Fracois Messier, Dr. Michael W. Miller, Dr. Michael D. Samuel, and Dr. Emmanuel Vanopdenbosch

CHRONIC WASTING DISEASE IN CANADIAN WILDLIFE: AN EXPERT OPINION ON THE EPIDEMIOLOGY AND RISKS TO WILD DEER

Prepared by:

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Dr. Trent Bollinger, CCWHC, SK, Canada
Dr. Peter Caley, CSIRO, Canberra, Australia
Dr. Evelyn Merrill, Univ. of Alberta, AB, Canada
Dr. François Messier, Chair, Univ. of Saskatchewan, SK, Canada
Dr. Michael W. Miller, Colorado Div. of Wildlife, CO, USA
Dr. Michael D. Samuel, Wisconsin Coop. Wildl. Res. Unit, WI, USA
Dr. Emmanuel Vanopdenbosch, Veterinary and Agrochemical Res. Centre, Belgium

Submitted to:

Canadian Cooperative Wildlife Health Centre
Western College of Veterinary Medicine
University of Saskatchewan
52 Campus Drive
Saskatoon, SK, S7N 5B4

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Final Report

Executive Summary

This document represents a summary of discussion, conclusions, and recommendations of an Expert Scientific Panel convened to: 1) provide a synopsis of chronic wasting disease (CWD) in free-living cervids in Canada, 2) evaluate the ecological and socio-economic implications of CWD in Canada, and 3) make recommendations on research and management actions to minimize and mitigate the effects of CWD in cervid species.

The emergence of chronic wasting disease, a transmissible spongiform encephalopathy potentially affecting mule deer, white-tailed deer and elk, is arguably the most important issue in the management of free-living cervids in North America. The disease has the potential to reduce cervid populations in the long-term, and to create major socio-economic impacts as observed in other areas in North America.

CWD has been detected in western Canada only recently, first in 1996 in farmed cervids and subsequently in 2000 in free-living cervids in Saskatchewan. Epidemiological investigations and surveillance programs of farmed cervids identified 40 game farms in Saskatchewan and 3 game farms in Alberta with the disease. CWD is thought to have been introduced into farmed cervids in Saskatchewan during the late 1980s by the importation of CWD-infected elk from South Dakota. Management programs to eradicate the disease in farmed cervids appear to have been successful and there are currently no known infected farms in Canada. Environmental contamination of some CWD-infected premises continues to pose a potential threat to wildlife. Of most significance, the presence of CWD in wild deer in some areas is a potential source of infection for farmed cervids and poses a continued threat to the long-term economical viability of cervid farming.

In Canada, CWD in free-living cervids appears restricted to three relatively distinct geographic foci in Saskatchewan, although surveillance efforts in many areas are inadequate to detect the disease at low prevalence. Hence, the disease may yet be detected in other areas. Intense, risk-based surveillance to determine the distribution of this disease should be a high priority over the next few years. Demonstration of a more widespread distribution of CWD within Saskatchewan or elsewhere in Canada would affect management response to this disease.

Results over the last two years in the Saskatchewan Landing area, Saskatchewan, indicate CWD is well established in the local mule deer population. In spite of initial attempts to reduce deer densities by increasing hunting harvest, deer densities in most areas of western Canada are more than sufficient to allow CWD to spread and increase in prevalence.

The range of species that may be infected with CWD is not known with certainty. Information from the USA would indicate all mule deer, white-tailed deer and elk are susceptible to the disease. Infection in moose has been recently confirmed experimentally, but similar data for caribou are not available. CWD does not appear to pose a risk to cattle or bison. The risk to humans appears to be extremely low. Nonetheless, the World Health Organization and other government health agencies recommend that any animals with a TSE disease not be consumed by humans.

The panel concludes that the emergence of CWD in free-living mule deer and white-tailed deer in Saskatchewan warrants an aggressive regional and national management and research response to prevent further spread of CWD and to control or eliminate the disease in wild cervids. The recent introduction of CWD in Canada, and its restricted distribution, provides us with a unique opportunity to manage CWD before it is too late.
Once established in a population of free-living cervids, control or eradication of CWD is extremely difficult. Preventing establishment of new foci of CWD should be given the highest priority, which entails preventing the movement of CWD-infected cervids and infectious material to new areas. To prevent natural spread from endemic areas, and to reduce potential environmental contamination with infectious prions, severe population reductions of deer, to levels of <1 animal/km^2 of critical habitat, will likely be required for at least a decade. Complete removal of deer in local areas may eliminate focal introductions of CWD. Deer densities that can prevent spread of CWD, and sizes of buffer zones to contain CWD, are largely unknown at this time. Management programs will need to be developed using a research framework, and updated as we learn about this disease.

Canada is at a critical juncture in its response to CWD in free-living cervids. The Panel recognizes the success of the federal CWD program for game farms and envisions a comparable investment in the management of CWD in wildlife. Significant investment in CWD management and research by federal and provincial governments, within a national framework, is required and urgent in order to develop an effective response to this emerging disease.

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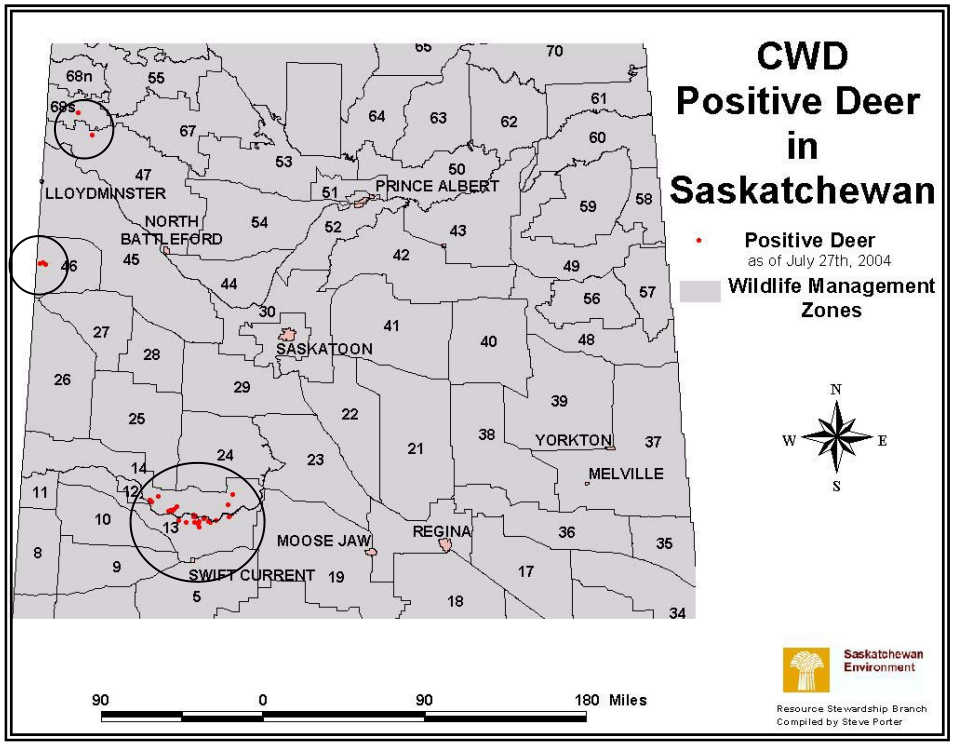
PREAMBLE

Chronic wasting disease (CWD) was first diagnosed in Saskatchewan in 1996 in a farm-raised elk (wapiti). In 2000, the disease was detected in a wild mule deer in Saskatchewan, and by the end of the 2003 hunting season, a total of 34 wild deer in Saskatchewan had been diagnosed with the CWD, the only wild deer populations in Canada thus far known to be affected with the disease. Affected animals have been detected at three relatively discrete geographic locations, but by far the greatest number (29) have come from the Saskatchewan Landing Area north of Swift Current (Map).



Chronic wasting disease is a newly-recognized disease of cervids with the potential to harm wild populations and to impose significant economic costs on Canadian society. Yet, it also is one of a group of diseases called transmissible spongiform encephalopathies, or TSEs, which are entirely new to science, and thus every aspect of CWD is shrouded in uncertainty. The Canadian Cooperative Wildlife Health Centre (CCWHC), an inter-agency partnership based at Canada's four colleges of veterinary medicine, has a mandate to provide sound scientific advice to its agency partners and to the public on important wildlife disease issues. In the face of the current new epidemic of CWD in wild cervids in Canada, the CCWHC assembled an international panel of scientists (Appendix 1) with the expertise required to evaluate CWD in Canadian wildlife and to recommend management, surveillance and research activities that would have the best chance of mitigating the full range of potential negative socioeconomic impacts associated with CWD in wild deer and elk in Canada. The occurrence of CWD in farmed cervids in Canada, and potential for transmission of CWD between farmed and wild cervids, was included in the panel's deliberations.

The Panel was asked to make full use of its collective expertise and the published scientific literature on CWD, on disease management, disease surveillance and the biology of North American cervids. Detailed information about the occurrence of CWD in Canada in both wild and domestic cervids was provided to the Panel in the form of written material from a range of Canadian sources (Appendices 2, 3 and 4). On 10-12 June, 2004, the Panel members assembled in Saskatoon. The Panel received information and questioned agency and other stakeholder representatives during an open forum on 10 June, and then prepared its report *in camera* on 11-12 June and through electronic exchanges thereafter.

The result of the Panel's deliberations is presented in this report. The Panel views CWD in Canadian wildlife to be a serious epidemic. The report outlines the nature and scale of the activities required to reduce the impact of CWD in Canada, and urges a coordinated national approach through which all relevant jurisdictions invest collectively in a unified program of management, research and mitigation.

Ted Leighton DVM, PhD
Executive Director
Canadian Cooperative Wildlife Health Centre
Headquarters Office, Saskatoon, SK

1. BACKGROUND

Chronic wasting disease (CWD) was first recognized in Canada in a herd of farmed elk (*Cervus elaphus*) in 1996. Further testing revealed that CWD was present on 40 game farms in Saskatchewan and three in Alberta. CWD is a reportable disease in Canada under the *Health of Animals Act*. Hence, an eradication program for CWD in farmed cervids was implemented in 2000 by the Canadian Food Inspection Agency. Results of eradication and surveillance activities in 2000-2004 support the view that successful eradication of CWD in farmed cervids is probable.

In wild deer populations, CWD was detected in mule deer (*Odocoileus hemionus*) in 2000, with confirmed cases in three discrete areas of Saskatchewan. For example, 21 mule deer with CWD were detected in a relative small zone in southern Saskatchewan (referenced hereafter as Saskatchewan Landing) during the hunting season of 2003. However, there have been no confirmed cases of CWD in wild deer populations within Canada outside of Saskatchewan.

The overall objective of the Panel is to provide an *expert opinion* on the best way to research and manage CWD in wild deer populations in Canada. We hope that our report will offer guidance to federal and provincial regulatory agencies in drafting policies to contain or eradicate CWD in free-ranging deer populations. A second but equally important objective of the Panel is to provide a package of information to the general public about risks associated with CWD based on data and experience gained internationally in the last decade or so.

In this report, the generic terms “deer species” or “cervids” refer to ungulate species and sub-species within the taxonomic family Cervidae.

2. MANDATE OF THE PANEL

- To improve collective understanding of CWD in Canadian wildlife.

- To review risk factors and implications of CWD to wild cervid populations, including future development of the disease throughout Canada.

- To provide an expert opinion on the potential risks of CWD to humans.

- To propose recommendations to manage impacts of CWD, focusing on surveillance and monitoring programs, prevention, eradication, containment, and human health.

- To encourage a National and International cooperative framework to assess risks and manage CWD in wild deer populations.

3. EPIDEMIOLOGY AND MANAGEMENT OF CWD

CWD belongs to a group of fatal, neurodegenerative disorders in humans and animals called transmissible spongiform encephalopathies or TSEs. Other TSEs include scrapie in sheep, bovine spongiform encephalopathy (also called "mad cow disease") in cattle, and Creutzfeldt-Jakob disease in humans. TSEs are thought to be caused by an abnormal form of proteinaceous agents called **prions** that are devoid of nucleic acid. Although CWD is not infectious in the classic sense, in practice it acts like an infectious agent. According to the prion hypothesis, infection occurs by conversion of normal prion proteins (PrP^{c}) into the disease-associated, misfolded form (PrP^{res}) that is highly resistant to degradation by proteolytic enzymes. Disease is characterized by slow accumulation of abnormal prions in lymphoid and nervous tissue. Clinical signs of the disease typically appear after >1.5 years, as accumulation of prions causes microscopic spongiform lesions in the brain. Animals in the later stages of the disease exhibit behavioral changes and progressive loss of body condition. The clinical signs of CWD are not unique however, and CWD can be confused with other diseases.There is no immune response produced in an affected host. Currently there are no treatments or vaccines for prion diseases, and all infections are believed fatal.

CWD is the only TSE agent that is transmissible in free-ranging cervid species, including elk, mule deer, and white-tailed deer (*Odocoileus virginianus*). The disease was initially recognized in Colorado and Wyoming, first in captive cervids in the 1960s and subsequently in free-ranging cervids in 1981. The actual length of time that CWD has been present in North American is unknown. Distribution of the disease in North America is largely unknown, because adequate sampling and surveillance have not been conducted in most areas of the continent. Currently, CWD is found in free-ranging cervids in portions of Colorado, Nebraska, South Dakota, Wyoming, Saskatchewan, New Mexico, Illinois, Utah, and Wisconsin.

Specific details regarding the transmission of CWD remain uncertain; however, in most respects CWD behaves like an infectious disease. Contact between infected and non-infected animals via saliva, urine, and feces are the most likely direct routes of transmission. Transmission via contact between susceptible and infectious individuals probably requires more than transient exposure. It is not known when an infected animal begins shedding disease-causing prions, but it likely occurs long before clinical signs of disease and may be progressive through the course of the disease. Studies on CWD transmission in captive deer and elk indicate that lateral transmission (i.e., among a group of potential hosts sharing a common environment) occurs by direct contact and ingestion of abnormal prions. Vertical transmission (i.e., from mother to offspring via placental transmission or milk) does not seem to be a major route of infection. Transmission occurs among susceptible cervid species and from infected cervids to the environment, then to susceptible animals. However, the mechanisms for direct or environmental routes of transmission and their relative importance in free-ranging cervids are not understood. Abnormal prion proteins have remarkable persistence in the environment and are highly resistant to a range of treatments that typically kill or inactivate conventional infectious agents. Because CWD is readily transmitted among captive deer and elk concentrated in pens, it is believed that transmission is facilitated by the concentration of animals related to artificial feeding and baiting. Relative susceptibility to transmission among cervids and for

other wildlife species has not been established. Unlike scrapie in sheep, research indicates that genetic resistance in deer and elk is unlikely; however, the potential for genetic influences on susceptibility remains under investigation.

Little is known about the rate of increase in prevalence and geographic spread of CWD or the factors that affect these rates. Increases in CWD prevalence and geographic spread in Colorado and Wyoming have been relatively slow. Epidemiological modeling suggests that prevalence in Colorado and Wyoming may have increased 0.5 to 0.7% annually during the 1980s and 1990s. In addition, CWD has increased in prevalence and in geographic spread throughout areas in Colorado, Wyoming, and Nebraska despite the relatively low density of cervids present in these areas (2-5 animals per km^2). Although uncertainty remains about the mechanism of CWD spread across landscapes, it is generally believed that dispersing animals are one likely avenue of disease spread. In addition, human activities, particularly translocation of captive and free-ranging animals, have resulted in CWD range expansions, and once established, the disease may be maintained through environmental contamination for an unknown period of time. Currently, there is no evidence that CWD will spontaneously disappear or be controlled without management intervention. In contrast, there is significant potential for expansion of the geographic range of the disease.

The likelihood of interspecies transmission of prion diseases is influenced by the degree of homology of the infective prion proteins (PrP^{res}) with that of the host prion protein (PrP^{c}), giving rise to the concept of a "species barrier" which must be overcome before an infective prion strain from one species causes disease in another species. In addition, different strains of prions may occur within one animal species. At present, research on biological strain typing involves a variety of methods including biological models using laboratory rodents, molecular, and immunohistocemistry (IHC) methods. *In vitro* conversion experiments indicate that CWD prions can convert human as well as bovine and sheep prion proteins into its abnormal conformer (PrP^{res}), albeit at a very low rate. However, this research is not conclusive because many other factors (e.g., dose, strain of the agent, route of exposure) may also determine the level of the species barrier. CWD has been experimentally transmitted after intracerebral inoculation to a number of animals, including cattle. However, cattle did not become infected when exposed orally to infective prion proteins specific to CWD. At present, it can be concluded that the species barrier may not completely protect other cervid species, including caribou and moose, from CWD.

Most cases of CWD in cervids are diagnosed by *post mortem* laboratory testing on lymphoid or brain tissues. Studies indicate that, compared to brain tissue, lymphoid tissue accumulates CWD prions at early stages of disease development in most cervid species. Thus, testing lymphoid tissue allows for earlier detection of disease. Current recommendations based upon the accumulation of CWD prions in cervid species include testing of retropharyngeal lymph node and brain obex (with intact dorsal motor nuclei of the vagus) for the diagnoses of CWD. *Ante mortem* diagnosis using tonsillar biopsies has also been used to detect CWD in live deer. Tonsillar biopsy also appears to be a valid method for detecting CWD during the incubation stage. Although tonsillar biopsy may be used as an *ante mortem* and pre-clinical diagnosis, this approach requires capture of live animals, is only suitable in limited situations, and is not generally recommended for CWD surveillance. Other *ante mortem* tests are currently under

investigation. However, diagnoses of CWD using *post mortem* tissues rely on classical TSE test methods of Western blot and immunohistochemistry as reference and confirmatory tests. Recently, additional high-throughput assays were licensed in the United States for diagnostic screening for CWD in three species of cervids. Only one of these tests has been evaluated satisfactorily in Canada.

4. ORIGINS OF CWD IN CANADIAN WILDLIFE

The origin of the prion strain that causes CWD in deer and elk remains unknown: whether CWD has always been a natural disease of native North American cervid species or is a new manifestation of another animal prion strain (e.g., scrapie) cannot be determined from available information, and may never be known with certainty. However, based on current distribution and prevalence of CWD in Canada and the USA, it appears most likely that CWD was recently introduced into free-living cervids. Consequently, the panel supports the management perspective that CWD was not present historically in free-living Canadian cervids, and thus that this disease is not part of native ecosystems.

Published accounts, historical records, and results of ongoing epidemiological investigations suggest that captive, CWD-infected deer and elk were likely imported into Canada from the USA at least twice over the last 30 years; although not reported, additional introductions seem plausible. The earliest incursion of CWD into Canada in the 1970s (or earlier) appears to have been confined to mule deer in a single zoo in Ontario, without further spread. The second incursion in the 1980s (or earlier) began on at least one game farm in Saskatchewan where infected elk had been imported, with subsequent spread among game farms. Because available epidemiological findings cannot explain fully all of the documented CWD outbreaks in captive deer and elk on Canadian game farms, other undocumented incursions and/or other sources of infection may have occurred in the last few decades.

The known foci of CWD in free-ranging deer in Saskatchewan are most likely a result of unintentional spill-over from infected game farms. As presently understood, the geographic pattern of CWD distribution in native deer suggests at least two independent spill-over events where CWD became established in local free-ranging populations: an infected game farm was almost certainly the source for one of these, and seems the most likely source for the other. Current knowledge supports the notion that CWD epidemics in free-ranging deer in Canada have spread geographically, and that CWD is well-established in at least one free-ranging deer population (Saskatchewan Landing). There appear to be no natural barriers to further spread of CWD in Canada.

5. POTENTIAL IMPACTS ON WILDLIFE POPULATIONS, HUNTING AND VIEWING OPPORTUNITIES, AND ASSOCIATED ECONOMIC REVENUES IN CANADA

To date, natural cases of CWD have been found only in mule deer, white-tailed deer, and Rocky Mountain elk (*Cervus elaphus nelsoni*), but it is likely that subspecies of these cervid species are also susceptible. Although no natural cases of CWD-affected caribou (*Rangifer tarandus*) or moose (*Alces alces*) have been reported, CWD recently has been induced experimentally in moose by ingestion of infected tissues. Susceptibility of caribou to CWD remains unknown, but some level of susceptibility seems likely based on the similarities between the normal cellular prion protein of caribou and the normal cellular prion protein of mule deer. Although current CWD surveillance programs in Canada target deer and elk, moose and caribou probably should not be ignored because dispersal behavior of moose, and large herd sizes, seasonal aggregations and range fidelity of caribou suggest a high potential for CWD to spread in Canada if it were to be introduced into either of these species.

Implications of CWD for wild populations remain unclear. The disease is fatal, and affected animals will invariably die because no known treatment or vaccine currently exists. Although time to death can vary from a few days to about a year in captive animals once clinical signs of CWD appear, time to death is probably shorter in free-living animals given the factors that affect the longevity of diseased animals in the wild. There is no current information to suggest that the disease strongly affects the overall dynamics of infected populations in the short term, but the disease has not been observed long enough to know the ultimate population effects. Modeling projections from data collected in Colorado suggest that mule deer populations at the center of the affected area may decline in 40-50 years. However, insights from the modeling efforts to date are hindered by an unclear idea of how the disease is transmitted, and an incomplete understanding of the relationships between transmission rates and factors such as population density and size, age and sex structure, degree of spatial aggregation, seasonal movements, and social organization. Key to understanding the effects of CWD on free-living cervids are host densities or spatial structures at which the disease can decline in prevalence, and movement patterns among infected populations that may foster geographic spread. If threshold densities for disease persistence are low, the host population will need to be severely reduced in order to restrict the spread of CWD, which may be logistically or politically infeasible. Complicating our understanding of the impact on CWD populations is the resilience of the CWD agent in the environment. Environmental contamination may allow the disease to persist even with substantial herd reductions. As we gain additional understanding of the factors that influence transmission, spatially explicit epidemiological models may offer further insights into the impacts of the disease and management approaches that can constrain its spread into new areas.

In the immediate future, local management responses to the presence of CWD seem more likely to influence the demography of affected herds than the disease itself. Limited ability to diagnose the infection in live animals, long incubation periods, subtle clinical signs, and the intensive sampling efforts required to detect the disease make it unlikely that CWD will be detected in free-living cervids prior to the point at which it can be eradicated without intensive control programs. As a result, where cases are detected, management goals are likely to focus

on preventing spread, and thus will include some form of intensive control of the population or segments of the population. Control efforts for cervid populations may range from selective removal of clinical suspects to localized reductions in areas of high CWD prevalence and/or adjacent buffer areas. Where the goal is less than 50% overall population reduction, populations would be expected to rebound in the short-term given normal population reproduction and influx of animals from surrounding areas. In addition to the direct effect on population sizes, intensive reduction programs could cause local shifts in animal distributions or alter movement patterns and migratory behavior. Such behavioral responses may have implications not only for the well being of the targeted and adjacent herds, but may produce new challenges arising from increased trans-jurisdictional movements, differing administrative mandates, and public interests. Further, limitations on baiting and feeding cervids for CWD management may have consequences for local changes in distribution and productivity of some individuals or herds.

Although herd reduction programs based on more liberal seasons and permits may initially increase hunting opportunities, the long-term fate of the hunting culture in the face of CWD is unknown. Initial observations in Canada and the USA indicate that the majority of hunters will continue to hunt in their traditional or preferred area and process cervid meat for eating even if CWD has been detected in the wild. However, if the prevalence of CWD becomes high, results of a public attitudes survey in Wisconsin indicate that hunters may abandon the sport. Alternatively, it is feasible that hunters will request certified testing for CWD on an individual animal basis. Where a diagnostic test is positive, the Wisconsin survey of public attitudes indicated that the majority of hunters would be concerned about eating the meat. Further, most governmental agencies currently advise against consuming CWD-infected meat. No government programs similar to those in the game farming industry currently exist to compensate hunters for destruction of infected meat or other animal products. In areas where management is focused on reducing the number of CWD-affected animals, these programs are incompatible with management of deer populations primarily for trophy hunting. Thus, in areas where CWD is relatively common, there is the potential that changing hunter attitudes may reduce the ability of managers to use harvest of cervids as an effective wildlife management tool. A loss of hunting participation also would result in a loss of revenues associated with the sale of hunting licenses, which would have far-reaching implications for a wide variety of wildlife programs at both the national and provincial/territorial levels. In 1996, Canadians spent over $800 million hunting wildlife with nearly two-thirds of these expenditures made by large game hunters. Ultimately, public perception about the safety of handling and consuming suspect meat in areas of endemic CWD, and the quality of the hunting experience in the face of eradication programs, may impact hunter participation in those areas and even in areas with no reported incidence of CWD.

In addition to hunting, cervids are enjoyed by wildlife viewers. Management programs directed at reducing free-living cervids infected with CWD, particularly those in or near provincial and national parks, are likely to reduce viewing opportunities and associated revenues.

Secondary effects on other wildlife species from CWD-based management of cervids are of concern but are difficult to predict. The most likely impacts include shifts in prey selection by predators (primarily wolves, cougars, coyotes or bears) and scavengers (e.g., corvids and eagles) and local shifts in animal-vehicle collisions, herbivory, and competition with livestock.

Changes in these impacts are unknown and likely will be difficult to monitor, given current resources.

6. IMPLICATIONS OF CWD IN FREE-LIVING CERVIDS FOR FARMED ANIMALS IN CANADA

The coexistence of CWD-affected populations of free-living cervids with free-ranging or winter-fed cattle on public and private lands is not likely to have a direct impact on the cattle industry because no cross-species transmission of CWD has been reported, nor is it believed likely at this time. Nevertheless, concerns over the evolving nature of the disease are likely to keep the attention of ranchers focused on the disease. Although bovine spongiform encephalopathy (BSE) is not causally connected to CWD, occasional cases of BSE in Canadian or US cattle will likely stimulate questions and some level of concern about cattle exposure to the CWD agent. Scrapie, a naturally occurring prion disease of domestic sheep and goats, occurs in both Canada and the USA and has been the focus of control programs in both countries. As with BSE, scrapie has not been causally connected to CWD and occurrence of CWD should not hinder scrapie control efforts in Canada. In light of strain and epidemiological similarities between scrapie in sheep and CWD in deer, however, relationships between scrapie and CWD warrant further investigation. Secondarily, reduction in free-living cervids for CWD management may lead to increased predation on free-ranging cattle and sheep.

In contrast, the potential reservoir of CWD in free-living cervids will likely have significant and far reaching impacts on the cervid farming industry. Expansion of the industry would be constrained because of potential contamination in areas of infected free-living animals. Costs associated with fencing of new or established farms would increase dramatically. Double fencing, fence heights of 10 feet or more, and increased fence inspection undoubtedly would be necessary to ensure no fence-line contact with infected animals or ingressions of free-living cervids into pens and farm facilities. Further, fencing at these standards would need to be maintained for an extended time even after decontamination of CWD infected farms and restocking with CWD-free animals. Because improved fencing and maintenance cannot guarantee farmed cervids are not subject to exposure, the game farm industry likely will be in jeopardy unless effective preventive treatments become available. Even with the development of vaccines and *ante mortem* tests, the additional logistical difficulties and costs associated with precautionary activities to prevent infectious spread from the wild will rise significantly. In addition, public perception, both nationally and internationally, of the risks associated with game farm meat, velvet, and other products produced in areas of infected free-living cervids will likely impact game farms despite precautionary measures. Currently, game farm products produced in Canada are exported to various countries. Based on experiences with CWD and other TSEs, it is likely that agricultural trade sanctions, like the current Korean ban of elk velvet from Canada, would contribute to making the game farming industry in Canada potentially unsustainable in the long-term if CWD were to become wide-spread in free-living cervids in Canada.

Maintaining game farms in the presence of CWD in free-living cervids will require greater commitment of resources from governmental agencies given current regulatory responsibilities

and the need to compensate game ranchers in cases of depopulation. At present, federal and provincial/territorial agencies jointly assume the costs associated with inspections, laboratory diagnostic tests, veterinary investigations, carcass disposal, depopulation, and site decontamination. The number of incidences when these services are required is likely to rise significantly in areas with infected free-living cervids.

7. IMPLICATIONS OF CWD IN FREE-LIVING CERVIDS FOR HUMAN HEALTH IN CANADA

The prion strain thought to cause CWD has not been linked to cases of human illness in either Canada or the USA, and consuming venison from areas where CWD is present does not appear to increase the likelihood of people contracting sporadic Creutzfeldt-Jakob disease (CJD; a human prion disease). Moreover, experimental studies have demonstrated a substantial molecular barrier to conversion of normal human prion proteins in the presence of CWD prion proteins. Such a response is similar to the molecular barriers to human prion protein conversions by the prion strains that cause scrapie or bovine spongiform encephalopathy (BSE). Despite the reassuring nature of the findings in studies of human health risks conducted to date, there is public concern about the implications of human exposure to CWD and other animal prion diseases. This concern, based on experiences with massive exposure of people to the BSE prion in the UK and other European countries that apparently led to about 150 cases of variant CJD, will likely influence public attitudes toward CWD for the foreseeable future. Regardless of how unlikely human illness arising from CWD exposure may be, the perception that CWD could be a human pathogen will shape public attitudes toward hunting and consuming deer and elk in areas where CWD occurs. The panel recognizes and supports international public health officials' recommendations against consuming any parts of animals known to be infected with a prion disease.

8. MANAGEMENT PERSPECTIVES

Management options and predicted outcomes.

CWD is the only TSE agent known to affect wild cervid populations. Whereas experience in managing or eradicating scrapie in domestic sheep can be applied to managing CWD in farmed deer and elk, there is no similar experience with TSEs in wild populations. States such as Colorado and Wisconsin have recently undertaken CWD management programs aimed at eradicating or minimizing spread of the disease in wild cervids. Although the prevalence (the proportion of the population that is affected) of CWD has been reduced in some areas, it is still not clear how best to manage the disease in wild populations. Results from these programs are still preliminary, but can be used to guide other management programs and predict outcomes.

Two characteristics of this disease make it particularly difficult to manage. First, empirical data indicate CWD transmission can occur at low deer densities; this attribute necessitates high levels of population reduction or complete removal of deer in order to eradicate the disease. Second, evidence indicates infectious prions persist in the environment for years. Therefore, in

areas with high levels of environmental contamination, deer densities must be maintained at low levels for at least 5 to 10 years in order to ensure the disease is not introduced from the environment into re-established deer populations. Due to these two characteristics, once CWD becomes established in wild populations, eradication of CWD is difficult with current management options.

As eradication of CWD is extremely difficult, preventing establishment of new foci of disease must be seen as the primary objective of any CWD management program. All measures should be taken to prevent movement of potentially CWD-infected cervids or infectious material to new areas. These measures should apply to both agricultural and non-agricultural environments.

Where CWD is already established in wild populations, the management objective should be to reduce the prevalence of CWD in the population in order to reduce levels of environmental contamination, to reduce the probability and rate of spread, and to "buy time" until new methodological approaches for eradication are available. In the Panel's view, current levels of population reduction in CWD-infected areas of Saskatchewan will not prevent the disease from increasing in prevalence and spreading over time.

Preliminary information from Wyoming and Colorado suggests that containment of CWD likely will require reducing cervid densities to well below 1-2 deer/km^2 of critical habitat (i.e., winter range) across large areas. The area managed for reduction should consist of the area in which the disease has been detected, the core, and a surrounding area, or buffer zone, where deer from the core are likely to migrate or disperse. The size of the buffer zone must be based on knowledge of local movements and should ensure that the vast majority of deer moving out of the core area will disperse to areas where deer densities are sufficiently low that the probability of disease transmission would be extremely low. Removal of females and mature males in areas of high infection rates appear to be specific strategies that could minimize spread. In addition, specific strategies to cull animals showing clinical signs and to cull dispersing animals (i.e., yearling bucks) also may help to reduce spread. These high levels of population reduction will need to be maintained until alternative strategies are available to eradicate the disease.

Surveillance programs around infected areas must be sufficient to detect CWD at extremely low levels in order to identify new foci of disease. Complete depopulation of deer in an area around these foci, or so called "sparks," has a higher probability of preventing establishment of the disease, if detected early. Establishment of new endemic areas of CWD with long-term management programs as described above is highly undesirable. Consequently, preventing spread and stamping out sparks should receive the highest priority.

Management programs should be seen as experiments and must be designed to monitor outcomes, such as changes in deer densities, alterations in the age structure of populations, changes in disease transmission rates, size of the affected area, changes in disease prevalence, etc. These monitoring programs must be consistent and long-term in order to determine which management strategies work and which do not. Although CWD management experiments are being implemented in other parts of North America, they need to be replicated in order to validate the results. The slow moving nature of the epidemic makes management "failure"

difficult to detect and therefore monitoring programs must be carefully designed and well funded. Management programs should be adaptive in order to take advantage of new information as it becomes available.

Current approaches to surveillance and risk assessments

The document "Surveillance strategies for detecting chronic wasting disease in free-ranging deer and elk: results of a CWD surveillance workshop, Madison, Wisconsin, December 10-12, 2002" provides an excellent overview of this topic. It is available at "http:// www.nwhc.usgs.gov/research/chronic_wasting/CWD_Surveillance_Strategies.pdf".

Current surveillance programs to detect CWD in wild cervids are primarily based on testing deer and elk harvested through hunting. In order to minimize the costs of CWD surveillance, wildlife or deer management zones with a perceived high risk of CWD are typically sampled more intensively; whereas, other low risk zones are sampled less intensively, if sampled at all. Classification of a zone as high risk is based on proximity to known cases of CWD in farmed or wild cervids, or proximity to game farms with a history of CWD. Although the risk factors for CWD are poorly understood, proximity to populations known to be infected with CWD is an obvious risk factor due to potential movement or dispersal of animals. Other risk factors such as degree of aggregation of cervids should be considered in developing surveillance programs. In some provinces, risk assessments have been completed but the results have not been adequately incorporated into surveillance programs. For example, Saskatchewan Environment proposed a surveillance program at the International CWD Workshop in Saskatoon, SK, August 2003, based on proximity to cases of CWD, density of critical deer habitat and levels of artificial feeding or baiting; however, this risk-based surveillance program has not been fully implemented. Ontario is using a clearly defined risk-based approach in its CWD surveillance program. The Panel strongly encourages such approaches.

In several provinces, targeted surveillance of deer and elk showing signs of wasting and/or neurological disease is being used as a relatively inexpensive method of surveillance in low risk zones. Although useful, this strategy has significant limitations, especially in areas of low human densities where the probability of detecting animals with clinical signs is low. Results from this type of surveillance alone should not be relied upon to determine the occurrence of CWD in an area; rather, this approach should be used as a supplement to other surveillance methods if the goal of surveillance is to demonstrate absence of disease or early detection.

Sample sizes for hunter surveillance programs are typically established to detect relatively low (e.g. 1%) prevalence of disease with 95% confidence within a wildlife or deer management zone. Areas smaller than a wildlife management zone are intensively sampled in some cases due to the perception that these smaller areas are at high risk of disease. Given the clustered distribution of CWD and its relatively slow rate of spread, sampling at smaller spatial scales is appropriate in many situations. A short-coming in most, if not all, of the surveillance programs is a lack of precise location information for all wild deer and elk tested for CWD. Wildlife or deer management zones are typically too large to estimate prevalence of disease or monitor the introduction and spread of disease in an area. Precise location information allows spatially explicit modeling of disease dynamics.

A detection threshold of 1% in areas adjacent to known CWD infected deer populations is insufficient if the goal is to detect newly established foci and attempt to eradicate sparks. Sample sizes to detect disease at levels below 1% are recommended in these areas and samples should be pooled for no more than 2-3 years in order to detect early spread of CWD into these areas. Unfortunately, required sample sizes for extremely low prevalence may exceed sustainable harvest levels or public support in areas where CWD is not known to already occur.

Detection-based sampling should target adult animals (i.e., one year or older) as they are more likely to have detectable accumulations of abnormal prion proteins if they are infected. However, for research on the epidemiology of CWD, or for specific management needs, testing of fawns can be useful.

Current and evolving methods of testing

Diagnostic test procedures for detecting abnormal prion proteins in sampled individuals are constantly improving. Initially, diagnosis of CWD was based on observing spongiform (i.e. "sponge-like") change in brain tissue with the light microscope. However, these changes are only observed in animals in later stages of the disease and therefore this method does not detect earlier preclinical cases. Immunohistochemical stains specific for abnormal prion proteins (PrP^{res}) greatly improve the sensitivity and specificity of tests for CWD and permit early detections of CWD. Infection trials in mule deer and white-tailed deer have shown that abnormal prion proteins accumulate first in tonsil and retropharyngeal lymph nodes, followed by deposition in the dorsal motor nucleus of the vagal nerve in the obex region of the brain. As the disease progresses, abnormal prion proteins are found in other areas of the brain stem as well. A similar pattern of disease progression is observed in elk, but whether this is consistent among individual elk is still under study.

Sensitivity of the test procedure is dependant on which tissues are tested; of the three tissues most commonly sampled (retropharyngeal lymph node, tonsil, and medulla oblongata at the obex), retropharyngeal lymph nodes are the most sensitive (i.e.tests performed on this tissue detect earlier preclinical cases) and obex (i.e brainstem) is the least sensitive. Surveillance programs should clearly state the testing procedures and the criteria used to classify an animal as "test negative". These criteria should be standardized and validated amongst laboratories. In the past, different criteria have been used to define an animal as test negative. Hence, caution should be used when interpreting historical surveillance results, especially results from different laboratories. Less sensitive tests reduce the probability of detecting CWD, and consequently negative results are less meaningful than they would be if more sensitive tests were used.

The new "rapid" CWD tests detect abnormal prion proteins in unfixed tissues by using Western blot (WB) or enzyme-linked immunosorbent assay (ELISA) techniques. The sensitivities of these tests are similarly dependant on which tissues are analyzed. Sensitivities and specificities of these tests for a particular surveillance program should be determined and clearly stated when presenting results. The rapid tests have a high sensitivity but lower specificity which leads to false positives. Immunohistochemistry has high sensitivity and specificity and is appropriate as

a secondary test in order to reject false positives identified via initial screening. Appropriate samples need to be collected to ensure that positives from rapid tests can be confirmed with immunohistochemistry. When CWD surveillance is based on testing only retropharyngeal lymph nodes, formalin fixed and frozen brain samples should still be collected from each animal in order to confirm infection in positive animals and allow for strain typing of the abnormal prion proteins. This information is needed to understand the epidemiology of CWD in wild populations.

9. INFORMATION AND MANAGEMENT NEEDS FOR CWD

There are substantial information gaps to be addressed before the potential impact of CWD on Canadian wildlife can be forecasted accurately, and effective management implemented accordingly.

In the short term (within 1-3 years), defining the extent of the current epidemic is a key priority, requiring surveillance for CWD in wild cervids be timely and of the highest sensitivity. This goal requires:

- Developing better spatially explicit risk assessments to improve the detection power of surveillance programs. This type of risk assessment has been developed previously [e.g. Saskatchewan Environment, Ontario Ministry of Natural Resources], however it needs to be fully implemented.
- Improving efficiency of surveillance by combining information among species and sources of information
- Improving current surveillance to include the location of CWD-negative as well as CWD-positive animals, for both free-ranging and captive herds.

Currently, short of total depopulation and/or wildlife barrier fencing, it is unknown what type of management intervention can reliably prevent the spatial spread of CWD in wild cervids. Quantitative modeling in combination with available data provides the best approach to exploring management scenarios.

- Models should build on existing models (e.g., Gross & Miller 2001), as well as critiques of such models (e.g., Schauber & Woolf 2003).
- Model selection should be empirical, incorporating the latest available information arising from research and management of CWD in cervids.
- Models to evaluate management interventions should be stochastic and be spatially explicit, including habitat-dependent movements of host animals.
- Model predictions of the threshold population density of hosts, or of management regimes, such as culling, that will lead to reduction in prevalence and spread of CWD and/or its eradication, should guide management actions and monitoring in an adaptive management framework. (It is recognized that population densities close to zero may be required)
- Models with the purpose of forecasting the impact of CWD on Canadian cervid populations, in the broader context, should include the interactions with large predators on disease dynamics.

Even under the best case scenarios of the current outbreak of CWD in mule deer in the Saskatchewan Landing area, preliminary modeling indicates substantial reductions in deer population density ($\leq$1 animal km^{-2} of critical habitat) will be required to have any chance of disease containment. This level of population reduction likely cannot be accomplished by recreational hunting alone. There is a need to:

- Determine the rates and patterns of disease transmission and spread in order to design effective control strategies.
- Obtain information on human dimensions and perceptions of CWD in Canada.
- Explore methods for achieving a rapid (<2-3 yrs) and substantial ($\leq$1 deer km^{-2}) reduction in the population density of cervids over large areas (>1000 km^2), and the public acceptability of which methods, if any, can meet these targets.

There is a great need to better define potential host species for CWD, and more importantly, which hosts are most important in maintenance of CWD in an area, either individually or in combination with other sympatric hosts. Research on transmission of CWD among species, including humans, livestock and other wildlife should include the following:

- Continue efforts to quantify the risks posed to humans from consuming meat from the carcasses of CWD-infected animals.
- Quantify both intra- and inter-specific transmission of CWD between moose and caribou.
- Multi-host models should be developed to quantify the contribution of various transmission pathways within and among cervid species.
- A target list of other wildlife of concern (e.g., bison, muskoxen) should be developed, and prioritized for research.

There are several gaps in knowledge that continue to hamper understanding and management of CWD. It would be extremely advantageous to:

- Develop a rapid and inexpensive *ante mortem* field test for CWD.
- Develop tests to detect and quantify environmental contamination by abnormal prion proteins (i.e., CWD agent).
- Determine whether strain variation exists and can be used to assist in determining the origin of disease, and tracking of disease spread.
- Better understand the routes and rates of direct and indirect transmission of CWD prions. This goal will require focal research studies in order to better predict CWD spread in wild cervid populations. Specifically, we need further studies to assess how population spatial structure, movement rates and other ecological factors influence the establishment and spread of CWD in wild cervid populations.

10. CONCLUSIONS

It is imperative that a national plan is developed for monitoring, managing and researching CWD in wild cervids in Canada. The panel wishes to highlight the following conclusions, herein presented in point form for ease of understanding:

- The panel views the CWD issue to be of national importance.
- Unless some concerted and effective management action is undertaken in the near future, CWD will become widespread with the potential for major consequences to wildlife, game farming, and a variety of socio-economic interests in Canada.
- The panel recognizes the success of the federal CWD program for game farms and recommends a comparable investment in the management of CWD in wildlife.
- Notwithstanding the provincial jurisdiction over wildlife management, the panel sees the need for federal assistance in developing a national program to manage CWD in collaboration with provincial jurisdictions.
- Eradication is a desirable goal but extremely difficult to achieve in wild populations given current knowledge, technologies, and resources.
- Achieving a low or negligible level of prevalence of CWD is an appropriate strategy to reduce transmission rates, reduce the potential for spread, and to minimize the amount of transmissible prions in the environment.
- The panel recognizes the core elements for managing and preventing the spread of CWD to include:
    - Implement comprehensive surveillance for CWD in wildlife and game farms.
    - Prevent transmission of CWD between free-living cervids and animals in game farms.
    - Avoid artificial animal concentrations (e.g., baiting and artificial feeding)
    - Conduct scientific investigations that guide management of CWD
    - Control populations of free-living cervids to achieve disease management objectives.
    - Develop policies and regulations for animal translocations and other activities to prevent the spread of CWD.
    - Conduct scientific investigations to understand the epidemiology of CWD in wildlife populations.
- Recognizing the uncertainties associated with CWD, managing agencies should adopt an adaptive management approach to incorporate new information as it becomes available.

11. RECOMMENDATIONS

The Panel feels that there is a sense of urgency in taking actions to contain or eradicate CWD in Canadian wild deer populations. The Panel members are unanimous in supporting the following recommendations; they are grouped in sections but presented in no particular order of priority.

A: Management of game farms

1. Develop and implement policies to prevent transmission of CWD between game farm facilities and wildlife. Actions should include:
    - Do not permit new game farms in infected areas.
    - Use double fencing in infected areas.
    - Ensure previously infected farms are not accessible by wild cervids for a minimum of 5 years.
    - Develop policies and regulations for animal translocations that may lead to spread of CWD.

2. Maintain current surveillance and management programs for CWD in farmed cervids.

3. Conduct additional retrospective epidemiological tracing of all farms for more comprehensive risk assessments in cooperation with US authorities.

4. Mandatory CWD testing of all cervid mortalities on game farms.

5. Mandatory participation in CFIA and provincial surveillance programs for CWD.

6. Any transportation permit should be approved by both the import and export authorities.

7. Share information on surveillance results and epidemiological investigations among agencies with jurisdictions over wildlife and game farm animals in a timely fashion.

B: Management of free-living cervids

1. Develop and implement policies to minimize artificial aggregations of free-living cervids to reduce transmission of CWD. Actions should include:
    - Prevent access to hay stacks, salt blocks, and artificial water sources by wildlife in high risk areas.
    - Ban baiting or artificial feeding for cervids in high risk areas.

2. Develop and evaluate management programs for reducing prevalence and spread of CWD in cervids by:
    - Eradicating "sparks" (i.e., new foci of infection) through local depopulation and intensification of monitoring in surrounding areas.
    - Controlling CWD in infected areas through population reduction to a target density of 1 cervid/km^2 in "critical" habitat (i.e., winter range) with reassessment based on surveillance results.

2. Monitoring and surveillance of CWD:
    - Develop and implement a risk-based surveillance program on a national scale, e.g., SK and ON models.
    - Implement an aggressive surveillance program in the next 1-3 years to

document the distribution of CWD in free-ranging cervids in Canada.

- To prevent the spread of CWD, collect sample sizes in areas adjacent to infected areas that would allow the detection of prevalence at a level of 0.5% (5 infected individuals per 1000) with a 95% confidence level. The window of sampling can be up to 3 years.
- Adopt standardized diagnostic testing procedures at the national level.

C: Research needs

1. Evaluate the distribution of abnormal prion proteins (PrP^{res}) specific to CWD in different body parts of infected animals, and its implication to infectivity within a context of pathogenesis.

2. Assess the potential for transmission of CWD within moose and caribou populations.

3. Design an integrated research program to quantify the contribution of various transmission pathways within and among cervid species.

4. Develop spatially explicit models of CWD transmission and spread to guide management actions and monitoring in an adaptive management framework.

5. Collaborate in development and evaluation of diagnostic epidemiological tools including *ante mortem* tests, strain typing and environmental detection of prions.

D: Communications

1. Expand communication tools about the CWD issues and programs, including regularly maintained and linked websites, fact sheets about CWD distribution, and media releases. The targeted clientele should be broad based, including landowners, scientists, hunters, consumers, etc.

12. ACKNOWLEDGEMENTS

We thank all presenters who gave us the benefit of their advice with regard to CWD in Canada. The Panel was impressed by the quality of the written and oral presentations during the public sessions. The submitted briefs presented a national context to the CWD issue in Canada, both in wild deer populations and in farmed animals, and were essential to the Panel's evaluation.

The Panel acknowledges the particular support of Ted Leighton, Marnie Paskaruk, Patrick Zimmer, and Jacqui Brown during public hearings and follow-up discussion. We express sincere thanks to the Canadian Cooperative Wildlife Health Centre for organizing this workshop and facilitating our work. Funding for the Panel was provided by the Canadian Wildlife Federation and by Environment Canada on behalf of the Canada Wildlife Directors Committee. Anna L. Leighton provided editorial assistance to improve the clarity of the text.

13. LITERATURE REFERENCED

Belay, E.D., R.A. Maddox, E.S. Williams, M.W. Miller, P. Gambetti, and L.B. Schonberger. 2004. Chronic wasting disease and potential transmission to humans. Emerging Infectious Diseases 10(6): 977-984. http://www.cdc.gov/ncidod/EID/vol10no6/03-1082.htm

Bishop, R.C. 2003. The economic effects of chronic wasting disease (CWD) in Wisconsin. Department of Agricultural and Applied Economics, University of Wisconsin-Madison, Staff Paper No. 463.

Bruce, M. E., A. Chree, I. McConnell, J. Foster, G. Pearson, and H. Fraser. 1994**.** Transmission of bovine spongiform encephalopathy and scrapie to mice: strain variation and the species barrier. Philosophical Transactions of the Royal Society of London B 343: 405-411.

Gross, J. E., and M. W. Miller. 2001. Chronic wasting disease in mule deer: disease dynamics and control. Journal of Wildlife Management 65: 205-215.

Hamir, A.N., R. C. Cutlip, J. M. Miller, E. S. Williams, M. J. Stack, M. W. Miller, K I. O'Rourke, and M. J. Chaplin. 2001. Preliminary findings on the experimental transmission of chronic wasting disease agent of mule deer to cattle. Journal of Veterinary Diagnostic Investigations 13: 91-96.

Johnson, C, J, Johnson, M. Clayton, D. McKenzie, and J. Aiken. 2003. Prion protein heterogeneity in free-ranging white-tailed deer within the chronic wasting disease affected region of Wisconsin. Journal of Wildlife Diseases 39: 576-581.

Menard, J., K. Jensen, and B.C. English. Projected economic impacts of a chronic wasting disease (CWD) outbreak in Tennessee. Agri-Industry Modeling and Analysis Group Industry Brief. May 2004 http://web.utk.edu/~aimag/pubs/CWD.pdf

Miller, M. W., and E. S. Williams. 2003. Horizontal prion transfer in mule deer. Nature 425: 35-36.

Miller, M. W., E. S. Williams, N. T. Hobbs, and L. L. Wolfe. 2004. Environmental sources of prion transmission in mule deer. Emerging Infectious Diseases 10: 1003̃1006.

Miller, M. W., E. S. Williams, C. W. McCarty, T. R. Spraker, T. J. Kreeger, C. T. Larsen, and E. T. Thorne. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming. Journal of Wildlife Diseases 36: 676-690.

National CWD Plan Implementation Committee. 2002. Implementation document for plan for assisting states, federal agencies and tribes in managing chronic wasting disease in wild and captive cervids. Nebraska Game and Parks Commission Draft 1.

O'Rourke, K. I., R.E. Besser, M. W. Miller, T. F. Cline, T. R. Spraker, A. L. Jenny, M. A. Wild, G. L. Zebarth, and E. S. Williams. 1999. PrP genotypes of captive free-ranging Rocky Mountain eld (*Cervus elaphus nelsoni*) with chronic wasting disease. Journal of General Virology 80: 2765-2769.

Petchenik, J. 2003. Chronic wasting disease in Wisconsin and the 2002 hunting season: gun deer hunters' first response. Bureau of Integrated Science Services Department of Natural Resources.

Peterson, M. J., M. D. Samuel, V. F. Nettles, Jr., G. Wobeser, and W. D. Hueston. 2002. Review of chronic wasting disease management policies and programs in Colorado. Colorado Wildlife Commission, Denver, Colorado, USA.

Priondata. 2002. Chronic wasting disease of deer: a review. June 11, 2002. UK. May 2004 http://www.priondata.org/data/A_CWDinfo.html

Prusiner S, B. 1991. Molecular biology of prion diseases. Science 252:1515-1522.

Raymond, G. J., A. Bossers, L. D. Raymond, K. I. O'Rourke, L. E. McHolland, P. K. Bryant, III, M. W. Miller, E. S. Williams, M. Smits, and D. Caughey. 2000. Evidence of a molecular barrier limiting susceptibility of humans, cattle and sheep to chronic wasting disease. The European Molecular Biology Organization Journal 19: 4425-4430.

Samuel, M.D., D.O. Joly, M.A. Wild, S.C. Wright, D.L. Otis, R.W. Werge, and M.W. Miller. 2003. Surveillance strategies for detecting chronic wasting disease in free-ranging deer and elk. Results of a CWD surveillance workshop. USGS-National Wildlife Health Center. May 2004 http://www.nwhc.usgs.gov/research/chronic_wasting/CWD_Surveillance_Strategies.pdf

Schauber, E. M., and A. Woolf. 2003. Chronic wasting disease in deer and elk: A critique of current models and their application. Wildlife Society Bulletin 31: 610-616.

Seidl, A., S.R. Koontz, M. Bruch, and L. Elder. 2003. Economic implications of chronic wasting disease. *In:* Agriculture and Resource Policy Report, Department of Agricultural and Resource Economics, Colorado State University, June 2003, APR03-07. May 2004 http://dare.agsci.colostate.edu/extention/pubs.html

Seidl, A., S.R., Koontz, L. Elder, and M. Bruch. 2003. Chronic wasting disease overview: hunter information. *In:* Agriculture and Resource Policy Report, Department of Agricultural and Resource Economics, Colorado State University, June 2003, APR03-04. May 2004 http://dare.agsci.colostate.edu/extention/pubs.html

Sigurdson, C. J., E. S. Williams, M. W. Miller, T. R. Spraker, K. L. O'Rourke, and E. A. Hoover. 1999. Oral transmission and early lymphoid tropism of chronic wasting disease PrP^{res} in mule deer fawns (*Odocoileus hemionus*). Journal of General Virology 80: 2757-

2764.

The Green Lane, Environment Canada. 2003. Expenditures on nature-related activities by Canadians *In:* Part A – Nature-related activities by Canadians in Canada. May 2004 http://www.ec.gc.ca/nature/DirectBenefits.htm

U.S. Department of Agriculture and U.S. Department of the Interior. 2002. Plan for assisting states, federal agencies, and tribes in managing chronic wasting disease in wild and captive cervids. Pp. 20.

U.S. Department of Agriculture and U.S. Department of the Interior. 2003. Progress report on the plan for assisting states, federal agencies and tribes in managing chronic wasting disease in wild and captive cervids (October 2002 – September 2003). Pp. 31.

Wild, M. A., T. R. Spraker, C. J. Sigurdson, K. I. O.Rourke, and M. W. Miller. 2002. Preclinical diagnosis of chronic wasting disease in captive mule deer (*Odocoileus hemionus*) and white-tailed deer (*Odocoileus virginianus*) using tonsillar biopsy. Journal of General Virology 83: 2617-2628.

Williams, E. S., and M. W. Miller. 2002. Chronic wasting disease in deer and elk in North America. Scientific and Technical Review of the Office International des Epizooties Aug 21 (2): 305-316.

Williams, E. S., M. W. Miller, and E. T. Thorne. 2002b. Chronic wasting disease: Implications and challenges for wildlife managers. Transactions of the North American Wildlife and Natural Resources Conference 67: in press

Williams, E. S., M. W. Miller, T. J. Kreeger, R. H. Kahn, and E. T. Thorne. 2002. Chronic wasting disease of deer and elk: a review with recommendations for management. Journal of Wildlife Management 66: 551-563.

Wolfe, L. L., M. M. Conner, T. H. Baker, V. J. Dreitz, K. P. Burnham, E. S. Williams, N. T. Hobbs, and M. W. Miller. 2002. Evaluation of antemortem sampling to estimate chronic wasting disease prevalence in free-ranging mule deer. Journal of Wildlife Management 66: 564-573.

Appendix 1: Panel members and affiliations

Dr. Messier is currently the Head of the Biology Department at the University of Saskatchewan. His major research interest is in population dynamics and ecology of mammals, specifically of polar bears in the Canadian arctic. His previous and current ecology research lab examines ecological questions on mammals ranging from Ord's Kangaroo rats, black-tailed prairie dogs, muskrat, skunks, raccoons, wolves, caribou, wood bison, grizzly bears, and polar bears.

Dr. François Messier (Chair)
Professor and Head
Department of Biology
University of Saskatchewan
112 Science Place
Saskatoon, SK, S7N 3G9
Canada
Email:francois.messier@usask.ca

Dr. Trent Bollinger is a wildlife veterinary pathologist and Director of the Western and Northern Region of the Canadian Cooperative Wildlife Health Centre (CCWHC) at the Western College of Veterinary Medicine in Saskatoon, Saskatchewan. He has coordinated the chronic wasting disease surveillance program for the CCWHC over the last 5 years. His research focuses on the pathology and epidemiology of disease in wildlife.

Dr. Trent Bollinger
Director Western/Northern Region
Canadian Cooperative Wildlife Health Centre
Department of Veterinary Pathology
Western College of Veterinary Medicine
University of Saskatchewan
52 Campus Drive
Saskatoon, SK, S7N 5B4
Canada
Email: trent.bollinger@usask.ca

Dr Peter Caley currently works as a research scientist for the Risk Analysis and Biosecurity Team at CSIRO Entomology, Canberra. He is a leading researcher into quantifying the role of wildlife as reservoirs of infectious diseases, particularly bovine tuberculosis. His professional research interests include vertebrate ecology, epidemiology of disease in wildlife, risk assessment of invasive species, vertebrate pest control, biological statistics and ecological modelling.

Dr. Peter Caley
CSIRO Entomology
GPO Box 1700
Canberra, ACT 2601
Australia
Email: peter.caley@csiro.au

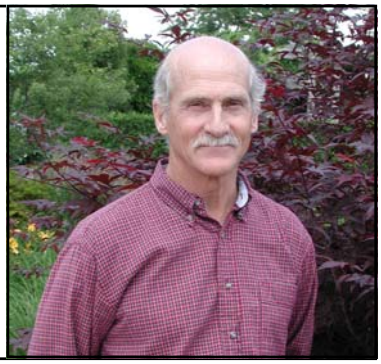
Dr. Evelyn H. Merrill is Associate Professor in the Department of Biological Sciences at the University of Alberta. She has conducted research on cervid foraging and habitat ecology for the past 20 years across ecosystems in Canada and the United States. Her current research focuses on cumulative impacts of industrial development and spatially explicit modeling of predator-prey dynamics in heterogeneous landscapes. She is a certified wildlife biologist and past associate editor for the Journal of Wildlife Management.

Dr. Evelyn Merrill
Associate Professor
Department of Biological Sciences
University of Alberta
B310 Biological Sciences Building
Edmonton, AB, T6G 2E9
Canada
Email: emerrill@ualberta.ca

Dr. Michael Miller has served as a wildlife veterinarian for the Colorado Division of Wildlife since 1989. In that capacity, he has researched a variety of topics related to the ecology and management of infectious diseases in free-ranging wildlife in Colorado and other states. Chronic wasting disease (CWD), the naturally-occurring prion disease of native North American cervids, has been the focus of a considerable part of Dr. Miller's research throughout the last decade. His studies, in collaboration with Dr. Elizabeth Williams and a host of other prion disease investigators, have included work on various aspects of CWD epidemiology, as well as on development and evaluation of surveillance systems and diagnostic tests used to detect and monitor infections in free-ranging populations of deer and elk. Dr. Miller is one of several collaborating scientists involved in surveillance-based adaptive management of CWD in North America, and has served in an advisory capacity to wildlife biologists designing surveillance programs to detect and contain new foci of CWD.

Dr. Michael W. Miller
Colorado Division of Wildlife
Wildlife Research Center
317 W Prospect Road
Ft. Collins, CO 80526-2097
USA
Email: mike.miller@state.co.us

Michael D. Samuel is the Assistant Unit Leader at the USGS Cooperative Wildlife Research Unit and Associate Professor in Wildlife Ecology, at the University of Wisconsin-Madison. He has conducted research on wildlife disease problems for the past 15 years. Mike has been working on chronic wasting disease in Wisconsin since the disease was detected in 2002. His current research studies on CWD involve spatial demographic evaluation of disease patterns in Wisconsin, evaluation of factors influencing transmission in white-tailed deer, surveillance programs for detecting CWD, transmission to other wildlife species, feeding and baiting of deer, and epidemiological modeling.



Dr. Michael D. Samuel
Wisconsin Cooperative Wildlife Research Unit
204 Russell Labs
1630 Linden Drive
University of Wisconsin – Madison
Madison, WI 53706
USA
Email: mdsamuel@wisc.edu

Dr. Emmanuel Vanopdenbosch joined the former National Institute for Veterinary Research, now called Veterinary and Agrochemical Research (VAR) Institute, at Brussels in 1977 where he specialized within the Department of Virology, in enzootic viral diseases of cattle, i.e. viral digestive, respiratory and reproductive disorders.
He became head of the Department of Virology and since 1998 head of the Department of Biocontrol, mainly in charge of TSE diagnosis and research, but also vaccine control, electron microscopy and histopathology.

Dr. Emmanuel Vanopdenbosch DVM, Lic
Head of Department of Biocontrol
Veterinary and Agrochemical Research Centre
VAR-CODA-CERVA
Groeselenberg 99
B-1180 Ukkel
BELGIUM
Email: emvan@var.fgov.be

Appendix 2: Presentations made to the Panel

Ron Lind-Saskatchewan Environment (SE)
Margo Pybus-Alberta Fish & Wildlife
Allan Preston-Manitoba Agriculture
Todd Shury-Parks Canada
Lynn Bates-Canadian Food Inspection Agency (CFIA)
Colin Maxwell-Canadian Wildlife Federation (CWF)
Joe Schmutz-Centre for Studies in Agriculture, Law and the Environment (CSALE)

Appendix 3: Briefs received by the Panel

Saskatchewan:
Saskatchewan Environment – Submitted by Ron Lind
Saskatchewan Agriculture and Food – Submitted by Rob Kerr
Saskatchewan Wildlife Federation – Submitted by Peter Schlivert
Saskatchewan Health – Submitted by Ross Findlater
Saskatchewan Stock Growers Assoc. – Submitted by Bern Rothwell

Alberta:
Alberta Fish & Wildlife – Submitted by Margo Pybus
Alberta Ag & Food – Submitted by Gerald Hauer
Alberta Wildlife Society – Submitted by Kirby Smith
Alberta Fish & Game Association – Submitted by Martin Sharren

Manitoba:
Manitoba Conservation – Submitted by Vince Crichton

Ontario:
Ontario Ministry of Natural Resources – Submitted by Brent Patterson
Ontario Ministry of Agriculture & Food – Submitted by Bob Wright

New Brunswick:
New Brunswick Department of Natural Resources – Submitted by Rod Cumberland

Northwest Territories:
Resources, Wildlife and Economic Development – Submitted by Brett Elkin

Federal Government Agencies:
Canadian Food Inspection Agency – Submitted by Lynn Bates
Parks Canada – Submitted by Todd Shury

Non-Governmental Agencies:
Centre for Studies in Agriculture, Law and Environment – Submitted by Joe Schmutz
Canadian Wildlife Federation – Submitted by Leigh Edgar

Appendix 4: Participants on the open forum

Ted Leighton (Chair) – Canadian Cooperative Wildlife Health Centre
Connie Argue - CFIA
Lynn Bates - CFIA
Ken McDaid - Fair Chase League
Leigh Edgar – Canadian Wildlife Federation
Brett Elkin – Resources, Wildlife and Economic Development, Northwest Territories
Gerald Hauer – Alberta Agriculture and Food
Wayne Lees - CFIA
Ron Lind - Saskatchewan Environment, Fish and Wildlife Branch
George Luterbach - CFIA
Colin Maxwell – Canadian Wildlife Federation
Ole Nielsen (Edmonton)
Allan Preston – Manitoba Agriculture
Margo Pybus - Alberta Fish and Wildlife Branch
Peter Schlivert – Saskatchewan Wildlife Federation
Joe Schmutz - Important Bird Areas (CSALE)
Todd Shury - Parks Canada
Cathy Soos – Western College of Veterinary Medicine
Brian Longworth - Saskatchewan Stock Growers Association Rep.
Murray Woodbury - Western College of Veterinary Medicine
Al Arsenault-Saskatchewan Environment, Fish and Wildlife Branch